Maternal and Fetal Well-being

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Pregnancy outcomes can be improved by following modern recommendations for personal health maintenance. Adequate caloric intake, reflected by a weight gain of about 10 to 12.3 kg (22 to 27 lb) for women of average build, is associated with the lowest rate of perinatal mortality. Maternal dietary protein supplementation should generally be avoided because it may be associated with low-birth-weight pregnancies. Abstinence from social drugs offers the greatest positive opportunity to modify the health of a fetus. Serious perinatal infection can be prevented by preconception immunization (rubella), food hygiene (toxoplasmosis) and attention to the expression of virus in the mother (herpes simplex). Available data do not correlate exercise programs begun before pregnancy and continued during pregnancy with adverse fetal effects. Athletic capacity need not diminish postpartum. Most employment may safely continue until delivery. Routine recommendations for prolonged maternal disability leaves are not medically warranted.

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Americans are caught up in a new wave of health consciousness. The importance of personal health maintenance is enhanced during pregnancy by virtue of the responsibility for a new life. Frequently coupled with health consciousness is consumer awareness that does not allow physicians to rest on tired dogma in response to new, or old, questions. On a daily basis, patients are confronted with concerns about diet and nutrition, drug exposure, perinatal infectious risks and the advisability of exercise and employment during pregnancy. Pregnancy outcome can be influenced substantially by the patterns of response to these concerns.

Diet and Nutrition

Energy Requirements (Calories)

During the past two decades, follow-up studies and controlled trials of nutritional supplementation have better clarified the influence of caloric restriction on pregnancy outcome. Food shortages during World War II, because of their severity and abrupt onset and resolution, became in effect a natural experiment on severe dietary restriction.[1] A famine was induced by a Nazi embargo during the 1944-1945 winter in the western urban regions of Holland. The famine lasted six months. Overall, at the height of the famine, mean birth weights were reduced about 300 grams. Pregnant women exposed to the famine throughout their entire third trimester had infants of considerably lower birth weights, whereas those pregnant women exposed to famine only during the first

27 weeks had infants with little reduction in birth weight. Proper caloric support and nutrition returned immediately after the embargo was lifted. Follow-up studies of 125,000 men 19 years later did not show an alteration in the mental performance of children born during the famine.[2] Because of the severity of the famine, it is not possible to generalize the birth-weight effects of famine to contemporary diets, which may lack certain recommended vitamins and minerals[3] but otherwise provide sufficient caloric support.

Subsequent experimental[4,5] and nonexperimental[6] caloric supplementation trials have indicated a beneficial birth-weight effect only for the pregnancies of women who are in a negative energy balance or who are underweight and by inference underfed at the time of their pregnancy. Net caloric supplementation of about 425 calories per day beginning during the 16th week of pregnancy resulted in a mean birth-weight increase of 224 grams in Gambian women during the wet season, a period of food shortage and high work load. During the dry season, a period of ample food and positive energy balance, the tendency was for a *negative* effect of supplementation on birth weight.[5] A similar experimental trial of Asian mothers at the Sorrento Maternity Hospital in Birmingham, England, yielded the same results.[4]

North American studies indicate that the apparent adverse effect of dietary supplementation to adequately fed mothers is probably greatest when the caloric supplementation is given as protein.[6-8] In an experimental trial of high-protein dietary

supplementation for low-income black women in New York City, the mean birth weight was decreased slightly with protein supplementation (40 grams of casein a day).[7] More important, and actually startling, was the significant excess of very premature births in the protein-supplement group (9%) versus the controls (5%).[7] Associated with the prematurity were increases in intrauterine growth retardation and neonatal mortality. The effect of the high-protein supplement was different for women who smoked. Birth weights of their infants were greater than for comparable smokers who did not receive protein supplementation. In the Sorrento study, birth weights were also lower among the adequately fed women who received high-protein caloric supplements. Moreover, nutritionally deprived rhesus monkeys given protein supplementation also have an excess of premature births[9]; as in the New York study,[7] the maternal weight gain produced by protein supplementation was not transmitted into birth weight.

Maternal Weight Gain

Measuring maternal weights throughout a pregnancy is a practical alternative to counting calories for estimating energy and nutrient availability to a fetus. Weight gain of a mother and her prepregnancy weight both have positive linear associations with the birth weight of her child.[10] On a theoretical basis, the expected maternal weight increment can be estimated from the physical alterations in the maternal and fetal compartments. Almost 7.3 kg (16 lb) of a typical (mean) weight gain come from the fetus, placenta, amniotic fluid and growth of the uterus and breasts; another 5 kg (11 lb) are from general growth of the mother's body and storage of nutrient reserves.[11] This is consistent with suggestions for healthy women by the Committee on Maternal Nutrition of the National Research Council for a 12.5-kg (27.5-lb) weight gain[11] and by the Committee on Nutrition of the American College of Obstetricians and Gynecologists for a 10- to 12.3-kg (22- to 27-lb) weight gain.[12] Generally, little weight gain occurs in the first trimester. The last two trimesters are usually accompanied by an average gain of 0.34 to 0.40 kg per week (0.75 to 0.88 lb per week).[13] One function of weight gain during pregnancy is to provide stores of energy (in the form of fat laid down under the influence of progesterone) that serve as a protective energy buffer for the fetus. Thus, emphasis need not be placed on a precise, absolute weight gain for each week of pregnancy. Instead, suggested weekly increments should be viewed as guidelines.[14]

Optimal weight gain during pregnancy is related to prepregnancy body build.[15] Consequently, the 10- to 12.3-kg recommendation is only applicable for women of average build. Optimal weight gain for overweight women is only about half of the optimal weight gain for very thin mothers (Figure 1). Regardless of body build, maternal weight gains of less than 6.8 kg (15 lb) are associated with increased perinatal mortality rates that result from a direct effect on the fetus and probably not from maternal acetonemia.[16] Pregnancy should never be a period of weight reduction, even in obese persons. Conversely, a weight gain of more than 14.6 kg (32 lb) is associated with an increase in perinatal mortality regardless of body build.[15] Within these wide weight guidelines, undue emphasis should not be directed to weight gain; instead, the long-standing recommendation, "eat to appetite,"[17] should be encouraged.
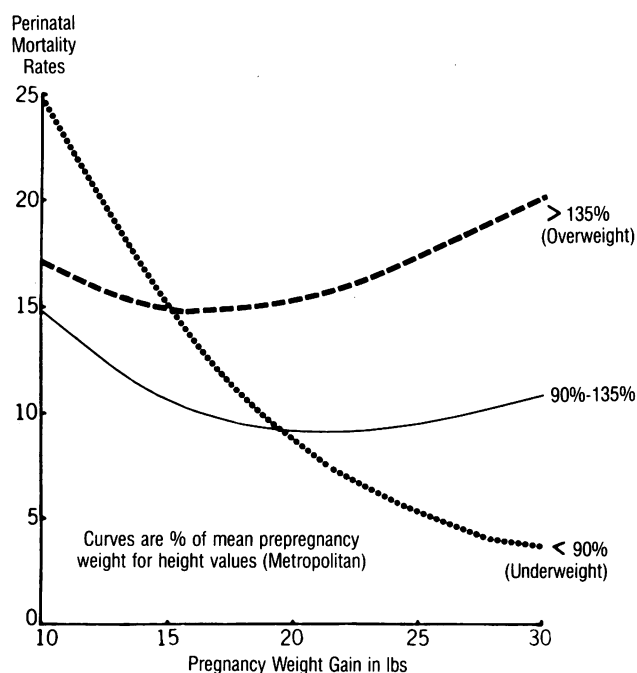

Figure 1.—Relationship of perinatal mortality rates to pregnancy weight gain according to maternal prepregnancy weight. (Perinatal mortality rates are expressed as infant deaths per 1,000 births.) (Modified from Naeye.[15])

Vitamins and Minerals

Opinion diverges regarding the value of vitamin and mineral supplementation during pregnancy. This results from different understandings of physiologic function during pregnancy. The physiologic changes of pregnancy are viewed either as adaptations to physiologic stress imposed by the fetus[18] or as adjustments to the process of fetal growth[11]—the differing views prompting different attitudes toward nutritional supplementation. "Adaptation to stress" implies the advisability of a nutritional response to an essentially undesirable situation. Alternatively, "adjustment to fetal growth" implies accommodation to an essentially normal process. The "stress" interpretation assumes obligate fetal requirements beyond those of a healthy nonpregnant woman. Yet it is clear that clinical standards held normal for nonpregnant women cannot be used as a standard for pregnant women. For example, plasma volume increases a maximum of 50% at the 34th week, the glomerular filtration rate increases about 60% and gastrointestinal motility generally decreases. These changes and the accompanying fall in serum vitamin and mineral concentrations may be viewed as a normal "adjustment" to pregnancy. This latter view is supported by the inability of experimental studies to detect a beneficial response of vitamin and mineral supplementation in pregnancy.[19]

Routinely supplementing iron to prevent anemia during pregnancy is widely advocated in the United States.[13, 20] Reassessment of this policy has been advocated by the World Health Organization[21] and others[11, 22]; new concerns have developed about high-dose iron supplementation and its potential association with intrauterine growth retardation.[23] During pregnancy, total erythrocytic volume rises an average of 250 ml over nonpregnant values. With supplemental iron throughout pregnancy, erythrocytic volume increases by 400

ml. Due to the more rapidly increasing plasma volume, mean hematocrits reach a nadir at 32 weeks' gestation. This fall is about 18% without iron supplements and is lessened to 13% with supplements.[13] To achieve maximum hemoglobin concentrations during pregnancy, total iron requirements are 600 to 800 mg of elemental iron.[13] This can be achieved by administering throughout pregnancy 30 to 60 mg a day of elemental iron.[3] Women who are known to have iron deficiency or who are at risk for anemia on the basis of poverty, adolescence or previous anemia should all receive iron supplementation.

Folate is required for not only the augmented maternal erythropoiesis of pregnancy, but also for DNA synthesis in the cells of all rapidly growing tissues such as the fetus and placenta. During normal pregnancy, serum folate levels decline progressively. This is partly due to increased urinary excretion. Although low serum folate levels (less than 3 ng per ml) occur in 20% to 25% of otherwise normal pregnancies, an associated megaloblastic anemia is uncommon in the United States.[24] Food sources encouraged during pregnancy should include those rich in folic acid, such as leafy vegetables, liver and, to some extent, nuts, cheese, eggs and milk. Because of folic acid's ready availability in the diet, many authorities stop short of recommending routine supplementation.[11-13, 22] Low-dose folic acid supplementation may reduce the incidence of neural tube defects in women who have had a prior child with a neural tube defect.[25,26] Before routine supplementation can be recommended, further confirmatory studies are necessary. When there is concern about a diet's adequacy for folic acid, a daily supplement of 800 µg is appropriate.[3,13]

Calcium metabolism adjusts during pregnancy to progressively promote maternal calcium retention. This begins during the first months of pregnancy, well before fetal skeletal mineralization, which begins in the third trimester. At term, a fetus stores about 27 grams of calcium, and maternal skeletal stores would be reduced by that amount if not replaced from dietary sources.[27] The recommended daily allowance of calcium during pregnancy is 1,200 mg, an increase of 400 mg over that for nonpregnant women.[3] Intake of calcium at this level should begin in the first trimester. Dairy products are the principal source of calcium in most diets. (One quart of milk contains exactly 1,200 mg of calcium.) Women who do not consume a similar amount of milk or dairy products will require supplementation. Vitamin D's importance in calcium metabolism is recognized, and one quart of vitamin D-fortified milk is sufficient to meet the recommended daily vitamin D requirements of pregnancy.[3]

The edema of pregnancy can be unpleasant but attempts to treat this condition with sodium restriction or diuretics are ill-advised, though common. Edema of pregnancy is physiologic, occurring in about 35% of normotensive women.[28] Salt restriction is not effective in managing preeclampsia, and diuretics have adverse effects.[29] Until the role of sodium in preeclampsia is clarified, pregnant women can use salt to taste.

Blood levels of most other vitamins and essential trace minerals are reduced during pregnancy, but whether this represents a deficiency or a physiologic change is speculative. Supplementation of these substances within the recommended dietary allowances[3] (for example, with the common prenatal multivitamin) probably is harmless and may be beneficial.[30]

Drug Exposure

Pregnant women want to know whether a drug will "hurt" their baby. Less concern relates to adverse maternal drug effects that have a specific relationship to pregnancy, such as tetracycline-induced acute fatty liver[31]; fortunately, such effects are rare. In spite of thousands of drugs available to physicians, relatively few have proven adverse fetal effects (Table 1).[32] Establishing the absolute safety of any drug during pregnancy is virtually an impossibility. Animal and in vitro study systems may yield results that are not applicable to humans. An uncommon occurrence with grave clinical results may not be detectable in human follow-up studies with fewer than many thousands of subjects. Studies such as these should be continued, but they are expensive and can be plagued with problems of bias. For these reasons, the standard for drug safety has become long-term observation of millions of women who have used the drug in question without ill-effects to their pregnancies. Decisions about other newer drugs often cannot be made with assurance because the data are insufficient. When in doubt, clinicians should consult existing drug catalogues.[33,34] Because of the difficulties in establishing safety, drug use during pregnancy should be limited whenever possible.

Interest should extend from prescription medications to social drugs such as alcohol, the most common proved human teratogen. Abstinence from social drugs offers pregnant women the greatest positive opportunity to modify their health and that of their fetus.

Alcohol

Maternal alcoholism has been clearly established as a severe risk for what is now known as the fetal alcohol syndrome.[35-37] It occurs in about half of the offspring of women with alcoholism, with an incidence in the United States of about 1 per 1,000 live births.[38,39] The principal anomalies include the following[40]:

- *Intrauterine growth retardation.* The pattern of growth retardation is generally symmetric, with proportional decreases in length, weight and head size that persist into infancy and probably into adult life with minimal potential for "catch-up growth."
- *Facial anomalies.* These are typically midfacial hypoplasia.
- *Mental retardation.* The IQs of children with the fetal alcohol syndrome have a broad range but seem to average about 70. In addition, minimal brain dysfunction with hyperactivity, decreased attention span and learning disabilities is common.[41]
- *Other.* Other associated anomalies have been reported, including cardiac septal defects and genitourinary anomalies.

While the severe risks of heavy alcohol consumption are definite, dangers from light drinking are less well established. The behavioral effects of moderate maternal alcohol consumption are difficult to measure in neonates and may only be apparent years later when schooling begins.[41]

Questions on drinking must be included in the routine evaluation. A physician's responsibility is to inform patients of the severe risks of heavy drinking and the potential danger of moderate drinking. Pregnant women with problems of heavy alcohol use should be encouraged to enter a treatment pro-

TABLE 1.—Major Effects of Maternal Drug Use on Offspring*

Drug	Major Effects on Offspring
Proved Human Teratogens†	
Thalidomide	Phocomelia
Phenytoin, trimethadione	Craniofacial and skeletal defects
Warfarinlike anticoagulants	Impaired nasal and optic development, epiphyseal stippling
Folic acid antagonists (aminopterin, methotrexate)	Cranial and skeletal defects
Androgens, some progestins	Masculinized genitalia (in female embryos)
Diethylstilbestrol	Genital tract abnormalities
Mercury	Central nervous system defects
Ethanol	Facial and central nervous system abnormalities
Suspected Human Teratogens‡	
Lithium carbonate	Cardiac defects (Ebstein's anomaly)
D-penicillamine	Connective tissue disorder (cutis laxa)
Benzodiazepines	Facial clefts
Oral contraceptive drugs	Limb and cardiac defects
Cancer chemotherapeutics	Varied effects
Nonteratogenic Adverse Drug Effects	
Aspirin	Premature closure of ductus arteriosus
Propranolol hydrochloride	Hypoglycemia, growth retardation
Thiazide diuretics	Thrombocytopenia, hypokalemia, hyponatremia, hyperbilirubinemia
Primidone, trimethadione	Depletion of vitamin K-dependent clotting factors
Chloramphenicol	Gray baby syndrome
Warfarin	Abnormal bleeding
Aminoglycosides	Auditory-vestibular nerve toxicity

*Modified from Scialli.[32]
†Unequivocal evidence links these drugs to human teratogenesis. While other drugs are probably teratogenic, supporting evidence is incomplete or lacking.
‡Data for the teratogenic effects of these drugs may be conflicting or incomplete. When more data are available, the teratogenic effects for these drugs may be proved.

gram to increase the likelihood that they will be able to abstain during pregnancy.[42]

Tobacco

The adverse effects of maternal smoking on human reproduction are extensive.[43,44] One of the most common neonatal findings in cases of maternal smoking is symmetric growth retardation.[45,46] These children remain smaller as adolescents and have significantly more behavioral problems, learning disabilities and poorer school performance.[47-49] Many other obstetrical complications are also increased in smokers. Included are selected congenital malformations,[50,51] abortion,[50,52] placental abruption and a substantial overall increase in perinatal mortality rates.[53] These untoward effects are produced, in part, by nicotine, which stimulates the sympathetic ganglia and releases catecholamines from peripheral nerve endings and the adrenal medulla. The increased maternal catecholamines reduce uterine blood flow that in conjunction with elevated levels of carboxyhemoglobin produces a fetal state of chronic oxygen deprivation and stress.[54,55] Indirect evidence suggests that the carcinogenic substances in tobacco smoke may act as transplacental carcinogens, increasing the liability of the offspring of smokers to tumors in adulthood.[56] Pregnant women should discontinue smoking at least for the duration of pregnancy.[57]

Caffeine

The Food and Drug Administration has recommended that pregnant women avoid or minimize intake of caffeine because animal studies indicate it causes birth defects in animals. This warning has been criticized because safe levels of caffeine were not defined,[58,59] and properly designed human studies have failed to show any adverse pregnancy effect associated with maternal coffee consumption.[60] In spite of the paucity of information on women showing adverse pregnancy effects of caffeine use, some pregnant women may wish to reduce their consumption. These women should be made aware of the significant amount of caffeine found in tea, cocoa and other foods and beverages.

Marijuana

Data regarding the impact of smoking marijuana during pregnancy are difficult to interpret due to the small size of the reported series. However, there are no findings to suggest that marijuana is teratogenic in animals or humans.[33(p269),61] Several studies have suggested that marijuana consumption during pregnancy leads to intrauterine growth retardation, premature labor, meconium staining, dysfunctional labor and features suggestive of the fetal alcohol syndrome.[62-64] However, lack of satisfactory control for potentially confounding factors (multiple drug use, socioeconomic status and the like) makes interpretation of these results difficult.[65] In mice, prenatal exposure to cannabinoids results in alterations of endocrine function and brain amine levels in adulthood. These effects, which are not evident at birth, become apparent only when the offspring themselves begin reproducing.[66,67] Unless substantial long-term studies exonerate maternal marijuana consumption from untoward effects on fetal growth and development, patients should be strongly advised against consumption during pregnancy.

Perinatal Infection

Concerns about the risks of infection are heightened for women during pregnancy. Although serum immunoglobulin

levels are decreased[68] and cell-mediated immune responses are suppressed in pregnancy,[69,70] the clinical significance of these findings remains conjectural, and pregnancy data do not support a substantially increased incidence of viral infection.[71] Nonetheless, the proportion of women with an infection that may have direct adverse consequences on a fetus is impressive, about 14% for the TORCH complex alone.[72] (TORCH is an acronym for *t*oxoplasmosis, *o*ther [syphilis, chickenpox, hepatitis B and the like], *r*ubella, *c*ytomegalovirus and *h*erpes simplex virus.) Further, approximately 15% of women are colonized with group B *Streptococcus*[73] and as many as 5% to 10% harbor *Chlamydia trachomatis* in the cervix.[74]

For members of the TORCH complex, the general measures for diagnosis and for maternal therapy are similar. The major health risk is confined to the fetus and infant. Maternal signs and symptoms that suggest infection should be immediately reported to a physician; symptoms alone are not reliable for diagnosis, however. Instead, maternal infection must be documented by serology or by viral isolation of the herpes simplex virus. Generally, accurate serologic diagnosis of current infection requires the finding of a significant rise in antibody titer. Serum should be obtained immediately when there is a suggestion of infection. A rise in antibody titer is usually found in a second sample obtained two to three weeks after onset of infection. Except for cases of syphilis and toxoplasmosis, maternal therapy is supportive with no special therapy available. If maternal infection is documented, at present, techniques are not available to diagnose the presence or absence of corresponding fetal infection. Amniocentesis for viral recovery is not predictive of an affected fetus. At birth, infant serology and culture will verify a microorganism, if present. Except for neonatal herpes simplex infections that produce high mortality and morbidity, infection with other TORCH agents does not generally result in apparent neonatal disease. However, late sequelae to these infections can result in damage to the perceptual organs and the brain, which would not be recognized until later in life.[72] Infectious control should be directed at prevention by immunization and minimizing exposure, if possible.

Maternal cytomegaloviruses, the most common cause of intrauterine infection, occur in about 13% of pregnancies[75] and 0.5% to 2.5% of live births.[76] Less than 5% of infected infants have classic cytomegalic inclusion disease (hepatosplenomegaly, microcephaly, chorioretinitis and so forth); however, subtle learning disorders and auditory deficits may be more common.[77] Much remains to be known about the means of transmitting cytomegalovirus between adults. The occupational contact of pediatric health-care workers does not appear to confer greater risks for infection.[78] At this time, screening for cytomegalovirus at the initial prenatal visit appears to be of negligible value.[79] The eventual development of an effective vaccine is the most promising avenue for reducing risk.

Toxoplasmosis, an infection caused by the protozoan parasite *Toxoplasma gondii*, is rare but potentially preventable during pregnancy. Congenital toxoplasmosis is associated with chorioretinitis, hydrocephalus and intracranial calcification plus a wide range of less severe manifestations.[72,80] Exposure to the parasite is by ingesting uncooked meat containing tissue cysts, or from oocysts in cat feces. Proper food handling and treatment of cat feces will avoid infection in

TABLE 2.—*Methods for Preventing Maternal Toxoplasmosis* *

Cook meat to ≥66°C (150°F), smoke it or cure it in brine
Avoid touching mucous membranes of the mouth and eye while handling raw meat
Wash hands thoroughly after handling raw meat
Wash kitchen surfaces that come into contact with raw meat
Wash fruits and vegetables before consumption
Prevent access of flies, cockroaches and so forth to fruits and vegetables
Avoid contact with or wear gloves when handling materials that are potentially contaminated with cat feces, such as cat-litter boxes, or when gardening
Disinfect cat-litter box for 5 minutes with nearly boiling water

*Modified from Wilson and Remington.[80]

susceptible women (Table 2). Routine serologic screening may prove to be efficacious in the future, but, at this time, quality control is apt to be haphazard and screening is not recommended.[80] For women known to have a primary infection during pregnancy, antimicrobial treatment appears to be effective in reducing fetal susceptibility.[81]

Herpes simplex virus acquisition by a fetus is rare (less than 0.05% of live births), occurring predominantly during birth through the vagina.[72] Disseminated and localized central nervous system disease affects most cases and severe central nervous system damage is common among survivors.

Pregnant women with a history of genital herpes or those with an undiagnosed genital lesion should notify their physicians. Whenever possible, in women without a history of genital herpes, viral cultures should be taken from all genital lesions. During pregnancy, suppression of recurring genital herpes with oral acyclovir is not indicated.[82] Women with recurrent genital herpes can expect the recurrences to increase as pregnancy progresses.[83] Asymptomatic viral shedding can occur from the cervix and vulva without a visible lesion.[84] This has prompted recommendations for viral screening cultures during the last month of pregnancy.[85] However, this is controversial because the cost is great and estimates are that with current techniques a maximum of only a fourth of infant infections could be averted.[86] At the onset of labor, all patients should have a careful inspection for herpetic lesions of the vulva, vagina and cervix. Because of the risk of asymptomatic viral shedding and ascending herpetic infection, vaginal examinations and instrumentation should be kept to a minimum in women with a history of genital herpes. Patients in labor who have a definite herpetic lesion (or an undiagnosed lesion suggestive of genital herpes simplex virus) should have a cesarean delivery, even in the absence of a positive viral culture, regardless of the duration since the rupture of membranes.

Cases of maternal and fetal rubella are rare, occurring annually in fewer than 50 infants in the United States. More than 30% of infants from pregnancies infected during the first trimester are seriously affected with a wide range of defects, including congenital deafness, cataracts and blindness and severe heart defects.[72] From 10% to 20% of reproductive-age women are seronegative for rubella antibodies. Seronegative, nonpregnant women of childbearing ages should be immunized and warned to avoid pregnancy for three months following vaccination.[87] Opportune settings include routine gynecologic and family planning facilities and during hospital stay after childbirth or abortion.

At present the recommendation is not to screen routinely for prenatal group B *Streptococcus* or *C trachomatis*. This relates to uncertainty regarding the proper timing for screening due to high maternal recolonization rates and the potential adverse effects from treatment of many pregnant women or infants who were colonized, but in whom disease would not have developed.

Exercise

Estimates are that 25 million Americans, or 10% of the population, jog regularly.[88] Pregnant women who jog or engage in other regular strenuous exercise are justifiably interested in its effects on their health and that of their fetus. Further, some competitors who become pregnant plan to continue strenuous sports and are interested in the effects that should be anticipated in their performance. Little is known about the effects of exercise on any of the specific pregnancy outcomes. However, insight into the potential consequences of exercise can be gained by considering its effects on maternal physiology. This subject has been recently reviewed by Lotgering and co-workers.[89]

Maternal oxygen consumption by organs other than the uterus is not increased substantially during pregnancy. Although maternal oxygen consumption near term increases by 16% to 32%,[90,91] most of the increase is related to uterine mass. Thus, exercise in the sitting position does not increase oxygen consumption more than with similar exercise in the nonpregnant state.[89] Because of the increased oxygen requirements of weight-bearing (the pregnant uterus) for treadmill exercise, however, increases in oxygen consumption are greater during pregnancy.[92] These data suggest that exercise efficiency is not altered substantially by pregnancy.

Blood flow to a pregnant uterus decreases immediately after the onset of exercise.[93,94] Decreases as great as 36% have been reported.[95] However, the implications of reduced oxygen availability to the fetus may not be valid. The hematocrit increases during exercise in association with a fall in plasma volume and increased glomerular filtration.[96] Further, in sheep, blood flow under the influence of catecholamines is redistributed from the myometrium to the placental site,[97] and a reduction in oxygen availability has not been noted.[95] Some studies of pregnant ewes[95,96] have indicated a significant fall in fetal oxygenation during maternal exercise. However, in these studies blood gas values were not corrected for the raised temperature that accompanies exercise.[89] In other studies in which a raised temperature was corrected for, only a small reduction in fetal oxygenation was found.[99]

Fetal heart rate studies have provided no consistent evidence of fetal distress during or after jogging or other exercise. Whereas some investigators noted modest fetal heart rate increases with maternal exercise,[100,101] others found fetal bradycardia with exercise.[102] Although exercise can induce fetal heart rate patterns that are consistent with fetal distress, the abnormal fetal heart rate patterns are not directly caused by the exercise. Instead, the exercise "unmasks" another condition such as nuchal cord,[103] placental insufficiency[103,104] and the like that has compromised the fetus. This has prompted some clinicians to suggest that the response of a fetal heart rate to maternal exercise could be used as a test of fetal well-being.[103,104] Hauth and associates detected no difference in the incidence of fetal heart rate abnormalities in

seven pregnant women after jogging on a total of 30 separate occasions.[105] At this time, limited studies do not support any adverse effect of maternal exercise on a fetus.[106]

Maternal exercise and physical fitness may be associated with a reduced duration of labor. Pomerance and colleagues found shorter labors for physically fit multigravid women, but not for fit nulliparous women.[107] Erkkola noted shorter spontaneous labors among women with above-average work capacity.[108] Collings and co-workers[101] detected no differences in the duration of labor of 12 women who engaged in a regular aerobic exercise program compared with 8 other women who did not exercise. The positive association between maternal exercise and shortened labor must be interpreted cautiously. Studies have not detected a reduction in the length of the second stage, that part of labor dominated by voluntary muscular activity which would appear most responsive to physical conditioning. Further, these studies did not control for analgesic use, which may be different in women who exercise.

Exercise conditioning can advance with training during pregnancy; postpartum, athletes should not expect a reduction in their athletic abilities. During pregnancy, exercise elicits the same metabolic and cardiovascular responses as it does in the nonpregnant state.[109] Erkkola noted a significant increase in the physical work capacity of randomly selected pregnant women who engaged in regular strenuous activity compared with nonexercising controls.[110] Olympic athletes who had had children judged their physical and functional condition to be better after childbirth than before pregnancy.[111]

Work

Newly pregnant women who are employed outside the home are confronted with a decision: Should I stop working before the end of the pregnancy and, if so, when? Considering that nonsalaried housekeeping and child rearing can be just as strenuous as other salaried employment, rational solutions are difficult. Available data and advice from physicians have been conflicting. Federal disability legislation has added new financial considerations that temper medical advice. This confusion is superimposed on prevalent societal expectations that have held sway for previous generations and vary widely across cultures.[112]

Available information is difficult to interpret for three reasons. First, there are considerable differences in the kinds of work women do. Second, studies carried out in different countries at different times would be expected to have different results. Third, employed women vary from nonemployed women in several aspects such as education, parity and the use of health services.[113]

More recent studies[114-116] have not supported an association between employment during pregnancy and adverse pregnancy outcomes that was found with earlier data.[117,118] Stewart noted more prematurity, low-birth-weight infants and perinatal deaths among the pregnancies of British women who were employed in Northamptonshire during their pregnancy.[117] If work ceased before 28 weeks' gestation, adverse outcomes were less frequent among the women who were employed. Naeye and Peters presented compelling data from the Collaborative Perinatal Project of the National Institute of Communicative Disorders and Stroke that suggested a significant progressive effect on birth weight by duration of employ-

ment when it continued past the 28th week of gestation.[118] The pattern of low birth weight suggested reduced uteroplacental blood flow. Murphy and associates had opposite results and different conclusions[115] using British data from the Cardiff Births Survey. Adverse pregnancy effects were all more common for nonemployed mothers, and the increase in premature births for prepregnancies of nonemployed mothers was statistically significant. Among employed mothers, the nature of the work (sedentary, nonsedentary) and time of stopping work did not significantly affect the results. A recent French national birth survey[113] had findings similar to the British study. Two hypotheses have been advanced to explain the superior pregnancy performance of the employed mothers in these two studies. First, by virtue of their ability to be employed, working mothers generally should be expected to be healthier and have less adverse pregnancy outcomes. Second, social contacts are enhanced for working women and their access to medical care is greater.

These relatively sparse and conflicting findings make it difficult to arrive at specific recommendations for the continuance of work during pregnancy. In addition to the medical consequences of work during pregnancy, the greater participation of women in the work force and passage of the Pregnancy Discrimination Act (PL 95-555) magnify the economic impact of physicians' recommendations about whether or not to continue employment during pregnancy.

The basic principle of the Pregnancy Discrimination Act is that pregnancy and related conditions must be treated in the same nondiscriminatory fashion as any other disability or medical condition. Basically, the law prohibits employers from illegally eliminating pregnant women from certain jobs and protects pregnant women's disability insurance benefits if the company has a disability plan and the pregnant worker is medically disabled. Because the law does not define disability or place time limits on the benefits, physicians will be asked to certify the extent and duration of the pregnancy-related disability.

As part of a physician's (and employer's) responsibility to minimize the effects of a pregnancy-related disability, every effort should be made to ensure that the specific job is safe and its environment is nontoxic to a mother and her fetus. When in doubt, regional consultants are available at the National Institute for Occupational Safety and Health (NIOSH) and the Occupational Safety and Health Administration (OSHA).[119]

The American College of Obstetricians and Gynecologists (ACOG) has drafted a principle applicable to most women during their pregnancies: "The normal woman with an uncomplicated pregnancy and a normal fetus in a job that presents no greater potential hazards than those encountered in normal daily life in the community may continue to work without interruption until the onset of labor and may resume working several weeks after an uncomplicated delivery."[119] The principle is consistent with the traditional six- to eight-week window of per se disability (beginning two weeks before delivery and continuing until six weeks' postpartum) that surrounds the time of delivery for most normal pregnancies.

Each situation should be interpreted individually. Guidelines for continuing various levels of work during pregnancy have been established by NIOSH and ACOG (Table 3).[120,121] For most employment, these guidelines support the concept of continued employment until delivery. Although this concept

TABLE 3.—*Guidelines for Continuing Various Levels of Work During Pregnancy**

Job Function	Week of Gestation
Secretarial and light clerical	40
Professional and managerial	40
Sitting with light tasks	
Prolonged (>4 h)	40
Intermittent	40
Standing	
Prolonged (>4 h)	24
Intermittent	
(>30 min/h)	32
(<30 min/h)	40
Stooping and bending below knee level	
Repetitive	
(>10 times/h)	20
Intermittent	
(<10 >2 times/h)	28
(<2 times/h)	40
Climbing	
Vertical ladders and poles	
Repetitive	
($\geq$4 times/8-h shift)	20
Intermittent	
(<4 times/8-h shift)	28
Stairs	
Repetitive	
($\geq$4 times/8-h shift)	28
Intermittent	
(<4 times/8-h shift)	40
Lifting	
Repetitive	
>23 kg (50 lb)	20
<23 >11 kg (25 lb)	24
<11 kg	40
Intermittent	
>23 kg	30
<23 >11 kg	40
<11 kg	40

*Modified from *The Journal of the American Medical Association* (1984:251:1995-1997), Copyright 1984, American Medical Association.

has been acknowledged by most large firms, this may not be the case in small companies and a physician may be thrust into a new role as patient advocate.[122]

Comment

Health maintenance in pregnancy extends beyond the highlights discussed in this article. Personal health maintenance, broadly interpreted, begins with preconceptual contraceptive planning and continues through to the choice of a site for delivery. The fundamental aspects of personal health maintenance are generally simple, but the effects of disease prevention and peace of mind may be substantial. Obstetricians should embrace these concepts to achieve optimal outcomes for their patients.

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